TABLE 4-2. SUMMARY OF EXPERIMENTS ON THE EFFECTS OF INHALATION OF ASBESTOS

Study	Animal species	Material administered	Dosage	Animals Examined for tumors	Findings (malignant tumors)	Average survival time
Gross et al. (1967)	132 male white rats	Ball- and hammer-milled Canadian chrysotile with/without 0.05 ml intratracheal 5 percent NaOH	42-146 mg/ml (mean concentration, 86 mg/m³) for 30 hours/week	72	17 adenocarcinomas 4 squamous-cell sarcomas 7 fibrosarcomas 1 mesothelioma	not available
	55 male white rats	Controls with/without 5 percent NaOH	control	39	none	not available
Reeves et al. (1971)	206 rats 106 rabbits 139 guinea pigs 214 hamsters	Ball-milled chrysotile, amosite, and crocidolite	48±2 mg/m³ for 16 hours/week up to 2 years	not available	2 squamous-cell carcinomas in 31 animals from crocidolite exposure	no information periodic sacrifices were made
Reeves et al. (1974)	219 rats 216 gerbils 100 mice 72 rabbits 108 guinea pigs	Ball- and hammer-milled chrysotile, amosite, and crocidolite	48±2 mg/m³ for 16 hours/week up to 2 years	120 rats 116 gerbils 10 mice 30 rabbits 43 guinea pigs	10 malignant tumors in rats, 2 in mice (Table 4-3)	no information periodic sacrifices were made
Wagner et al. (1974)	13 groups of approximately 50, and 15 of about 25 Wistar SPF rats	Amosite, anthophyllite, crocidolite, Canadian chrysotile, Rhodesian chrysotile (UICC samples)	10.1 to 14.7 mg/m³ for 1 day to 24 months, 35 hours/week	849	(See Tables 4-4 and 4-5) All asbestos varieties produced mesotheliomas and lung cancer, some from exposure as short as 1 day	669 to 857 days versus 754 to 803 for controls. Survival times not significantly affected by exposure.
Wagner et al. (1977)	CD Wistar male and female rats	Superfine chrysotile	10.8 mg/m³ 37.5 hours/week for 3, 6, or 12 months		1 adenocarcinoma of the lung in 24 animals exposed for 12 months	
	CD Wistar male and female rats	Nonfibrous cosmetic talc			none	
Davis et al. (1978)	46 groups of approximately 20 Han SPF rats and 20 Han SFP rats	UICC samples of amosite, chrysotile, and crocidolite	2 mg/m³ and 10 mg/m³ 35 hours/week for 224 days	208	7 adenocarcinomas 3 squamous-cell sarcomas, 1 pleural mesothelioma, 1 peritoneal mesothelioma	not available sacrificed at 29 months
	20 Han SPF rats	control	control	20	none	

TABLE 4-3. EXPERIMENTAL INHALATION CARCINOGENESIS IN RATS AND MICE

Fiber	Exposure[a] Mass mg/m³	Exposure[a] Fiber f/ml	Rats Animals examined	Rats Malignant tumors	Mice Animals examined	Mice Malignant tumors
Chrysotile	47.9	54	43	1 lung papillary carcinoma 1 lung squamous-cell carcinoma 1 pleural mesothelioma	19	None
Amosite	48.6	864	46	2 pleural mesotheliomas	17	None
Crocidolite	50.2	1,105	46	3 squamous-cell carcinomas 1 adenocarcinoma 1 papillary carcinoma - all of the lung	18	2 papillary carcinomas of bronchus
Controls			5	None	6	1 papillary carcinoma of bronchus

[a]The asbestos was comminuted by vigorous milling, after which 0.08 to 1.82% of the airborne mass was of fibrous morphology (3:1 aspect ratio) by light microscopy.

Source: Reeves et al. (1974).

TABLE 4-4. NUMBER OF RATS WITH LUNG TUMORS OR MESOTHELIOMAS AFTER EXPOSURE TO VARIOUS FORMS OF ASBESTOS THROUGH INHALATION

Form of Asbestos	Number of animals	Adenocarcinomas	Squamous-cell carcinomas	Mesotheliomas
Amosite	146	5	6	1
Anthophyllite	145	8	8	2
Crocidolite	141	7	9	4
Chrysotile (Canadian)	137	11	6	4
Chrysotile (Rhodesian)	144	19	11	0
None	126	0	0	0

Source: Wagner et al. (1974)

TABLE 4-5. NUMBER OF RATS WITH LUNG TUMORS OR MESOTHELIOMAS AFTER VARIOUS LENGTHS OF EXPOSURE TO VARIOUS FORMS OF ASBESTOS THROUGH INHALATION

Length of exposure	Number of animals tested	Number of animals with lung carcinomas	Number of animals with pleural mesotheliomas	Percent of animals with tumors
None	126	0	0	0.0
1 day	219	3[a]	2[b]	2.3
3 months	180	8	1	5.0
6 months	90	7	0	7.8
12 months	129	35	6	31.8
24 months	95	37	2	41.0

[a]Two rats exposed to chrysotile and one to crocidolite.

[b]One rat exposed to amosite and one to crocidolite.

Source: Wagner et al. (1974).

and female rats. No malignancies were observed in control groups of 60 males and females. The incidence of malignancy at other sites varied little from that of the controls. The authors note that if controls from other experiments in which ovarian and genitourinary tumors were present are included, the comparative incidence in the exposed groups in the first study lacks statistical significance. No data are provided on the variation of tumor incidence at extrapulmonary sites with asbestos dosage.

Wagner et al. (1977) also compared the effects of inhalation of a superfine chrysotile to the effects of inhalation of a pure nonfibrous talc. One adenocarcinoma was found in 24 rats exposed to 10.8 mg/m^3 of chrysotile for 37.5 hours a week for 12 months.

In a study similar to Wagner's, Davis et al. (1978) exposed rats to 2.0 or 10.0 mg/m^3 of chrysotile, crocidolite, and amosite (equivalent to 430 to 1950 f/ml). Adenocarcinomas and squamous-cell carcinomas were observed in chrysotile exposures, but not in crocidolite or amosite exposures (Table 4-6). One pleural mesothelioma was observed with crocidolite exposure, and extrapulmonary neoplasms included a peritoneal mesothelioma. A relatively large number of peritoneal connective tissue malgnancies also were observed, these including a leimyofibroma on the wall of the small intestine. The meaning of these tumors is unclear.

TABLE 4-6. EXPERIMENTAL INHALATION CARCINOGENESIS IN RATS

	Exposure		Number of animals examined	Malignant tumors
	Mass mg/m^3	Fiber f>5μm/ml		
Chrysotile	10	1,950	40	6 adenocarcinomas 2 squamous-cell carcinomas
Chrysotile	2	390	42	1 squamous-cell carcinoma 1 peritoneal mesothelioma
Amosite	10	550	43	None
Crocidolite	10	860	40	None
Crocidolite	5	430	43	1 pleural mesothelioma
Control			20	None

Source: Davis et al. (1978).

Inhalation exposures result in concomitant GI exposures from the asbestos that is swallowed after clearance from the bronchial tree. Although all inhalation experiments focus on thoracic tumors, those of Wagner et al. (1974), Davis et al. (1978), and, to a limited extent, Gross et al. (1967) also include a search for tumors at extrathoracic sites. A limited number of these tumors were found, but no association could be made with asbestos exposure.

One important aspect of the inhalation experiments is the number of pulmonary neoplasms that can be produced by inhalation in the rat as compared to other species (Reeves et al., 1971, 1974). This phenomenon illustrates the variability of species response to asbestos and the need for an appropriate model before extrapolations to man can be made with confidence. The absence of significant GI malignancy from asbestos exposure in animals, in contrast to that found in humans, may be the result of the use of inappropriate animal models.

4.6 INTRAPLEURAL ADMINISTRATION

Evidence that intrapleural administration of asbestos results in mesothelioma was presented in 1970 when Donna (1970) produced mesotheliomas in Sprague-Dawley rats treated with a single dose of 67 mg of chrysotile, amosite, or crocidolite. Reeves et al. (1971) produced mesothelial tumors in rats (1 of 3 with crocidolite and 2 of 12 with chrysotile) by intrapleural injection of 10 mg of asbestos. Two of 13 rabbits injected with 16 mg of crocidolite developed mesotheliomas.

In a series of experiments, Stanton and Wrench (1972) demonstrated that major commercial varieties of asbestos, as well as various other fibers, produce mesotheliomas in as many as 75 percent of animals into which material had been surgically implanted onto the pleural surface. The authors conclude that the carcinogenicity of asbestos and other fibers is strongly related to their physical size; fibers that have a diameter of less than 3 μm are carcinogenic and those that have a larger diameter are not carcinogenic. Further, samples treated by grinding in a ball mill to produce shorter length fibers are less likely to produce tumors. Although the authors attribute the reduced carcinogenicity to a shorter fiber length, the question was raised of the effect of the destruction of crystallinity, and perhaps other changes in the fibers, caused by the extensive ball milling (Langer et al., 1978).

Since 1972, Stanton and his co-workers (Stanton et al., 1977, 1981) have continued these investigations of the carcinogenic action of durable fibers. Table 4-7 summarizes the results of 72 different experiments. In their analyses, Stanton et al. (1981) suggest that the best measure of carcinogenic potential is the number of fibers that measure $\leq$0.25 μm in diameter and $\geq$8 μm in length, although a good correlation of carcinogenicity is also obtained for fibers $\leq$1.5 μm in diameter and $\geq$4 μm in length. The logit distribution of tumor incidence against the log of the number of particles having a diameter $\leq$0.25 μm and length $\geq$8 μm is shown in Figure 4-4. The regression equation for the dotted line is

$$\ln[p/(1-p)] = -2.62 + 0.93 \log x \qquad (4\text{-}1)$$

where p is the tumor probability and x is the number of particles per μg that are $\leq$0.25 μm diameter and $\geq$8 μm long. A reasonable relationship exists between the equation and available data, but substantial discrepancies suggest the possibility that other relationships may better fit the data. Bertrand and Pezerat (1980) suggested that carcinogenicity may correlate as well with the ratio of length to width (aspect ratio).

Another comprehensive set of experiments was conducted by Wagner et al. (1973, 1977). Mesothelioma was produced from intrapleural administration of asbestos to CD Wistar rats, demonstrating that there is a strong dose-response relationship. Tables 4-8 and 4-9 list the results of these experiments.

Pylev and Shabad (1973) and Shabad et al. (1974) reported mesotheliomas in 18 of 48 rats and in 31 of 67 rats injected with 3 doses of 20 mg of Russian chrysotile. Other experiments by Smith and Hubert (1974) produced mesotheliomas in hamsters injected with 10-25 mg of chrysotile, 10 mg of amosite or anthophyllite, and 1-10 mg of crocidolite.

Various suggestions have been made that the natural oils and waxes contaminating asbestos fibers might be related to the carcinogenicity of asbestos fibers (Harington, 1962; Harington and Roe, 1965; Commins and Gibbs, 1969). However, this theory was not substantiated in the experiments performed by Wagner et al. (1973) or Stanton and Wrench (1972).

TABLE 4-7. SUMMARY OF 172 EXPERIMENTS WITH DIFFERENT FIBROUS MATERIALS

Experiment	Compound	Actual tumor incidence	Percent tumor probability ± SD	Common log fibers/μg ≤0.25 μm diameter x ≥8 μm long	Experiment	Compound	Actual tumor incidence	Percent tumor probability ± SD	Common log fibers/μg ≤0.25 μm diameter x ≥8 μm long
1	Titanate 1	21/29	95±4.7	4.94	37	Halley 1	4/25	20±9.0	0
2	Titanate 2	20/29	100	4.70	38	Halley 2	5/28	23±9.3	0
3	Silcarbide	17/26	100	5.15	39	Glass 8	3/26	19±10.3	3.01
4	Dawson 5	26/29	100	4.94	40	Crocid 11	4/29	19±8.5	0
5	Tremolite 1	22/28	100	3.14	41	Glass 19	2/28	15±9.0	0
6	Tremolite 2	21/28	100	2.84	42	Glass 9	2/28	14±9.4	1.84
7	Dawson 1	20/25	95±4.8	4.66	43	Alumin 6	2/28	13±8.8	0.82
8	Crocid 1	18/27	94±6.0	5.21	44	Dawson 6	1/30	13±6.9	0
9	Crocid 2	17/24	93±6.5	4.30	45	Dawson 2	2/27	12±7.9	0
10	Crocid 3	15/23	93±6.9	5.01	46	Wollaston 2	2/25	12±8.0	0
11	Amosite	14/25	93±7.1	3.53	47	Crocid 12	2/27	10±7.0	3.73
12	Crocid 4	15/24	86±9.0	5.13	48	Attapul 2	2/29	11±7.5	0
13	Glass 1	9/17	85±13.2	5.16	49	Glass 10	2/27	8±5.6	0
14	Crocid 5	14/29	78±10.8	3.29	50	Glass 11	1/27	6±5.5	0
15	Glass 2	12/31	77±16.6	4.29	51	Titanate 3	1/28	8±8.0	0
16	Glass 3	20/29	74±8.5	3.59	52	Attapul 1	2/29	8±5.3	0
17	Glass 4	18/29	71±9.1	4.02	53	Talc 1	1/26	7±6.9	0
18	Alumin 1	15/24	70±10.2	3.63	54	Glass 12	1/25	7±5.4	0
19	Glass 5	16/25	69±9.6	3.00	55	Glass 13	1/27	6±5.7	0
20	Dawson 7	16/30	68±9.8	4.71	56	Glass 14	1/25	6±5.5	0
21	Dawson 4	11/26	66±12.2	4.01	57	Glass 15	1/24	6±5.9	1.30
22	Dawson 3	9/24	66±13.4	5.73	58	Alumin 7	1/25	5±5.1	0
23	Glass 6	7/22	64±17.7	4.01	59	Glass 16	1/29	5±4.4	0
24	Crocid 6	9/27	63±13.9	4.60	60	Talc 3	1/29	4±4.3	0
25	Crocid 7	11/26	56±11.7	2.65	61	Talc 2	1/30	4±3.8	0
26	Crocid 8	8/25	53±12.9	0	62	Talc 4	1/29	5±4.9	0
27	Alumin 2	8/27	44±11.7	2.95	63	Alumin 8	1/28	3±3.4	0
28	Alumin 3	9/27	41±10.5	2.47	64	Glass 21	2/47	6±4.4	0
29	Crocid 9	8/27	33±9.8	4.25	65	Glass 22	1/45	2±2.3	0
30	Wollaston 1	5/20	31±12.5	0	66	Glass 17	0/28	0	0
31	Alumin 4	4/25	28±12.0	2.60	67	Glass 18	0/115	0	0
32	Crocid 10	6/29	37±13.5	3.09	68	Crocid 13	0/29	0	0
33	Alumin 5	4/22	22±9.8	3.73	69	Wollaston 4	0/24	0	0
34	Glass 20	4/25	22±10.0	0	70	Talc 5	0/30	0	0
35	Glass 7	5/28	21±8.7	2.50	71	Talc 6	0/30	0	3.30
36	Wollaston 3	3/21	19±10.5	0	72	Talc 7	0/29	0	0

SD = Standard deviation.

Source: Stanton et al. (1981).

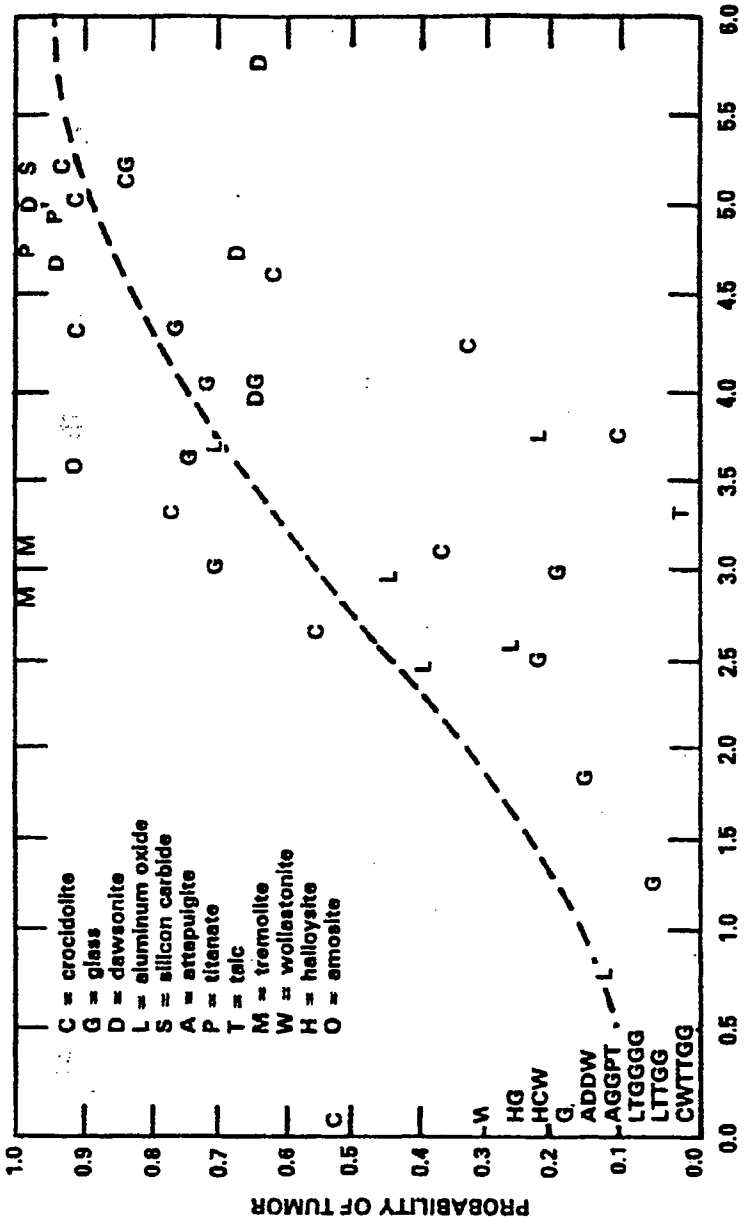

Figure 4-4.Regression curve relating probability of tumor to logarithm of number of particles per μg with diameter ≤ 0.25 μm and length >8 μm.

Source: Stanton et al. (1981).

TABLE 4-8. PERCENTAGE OF RATS DEVELOPING MESOTHELIOMAS AFTER INTRAPLEURAL ADMINISTRATION OF VARIOUS MATERIALS

Material	Percent of rats with mesotheliomas
SFA chrysotile (superfine Canadian sample)	66
UICC crocidolite	61
UICC amosite	36
UICC anthophyllite	34
UICC chrysotile (Canadian)	30
UICC chrysotile (Rhodesian)	19
Fine glass fiber (code 100), median diameter = 0.12 μm	12
Ceramic fiber, diameter = 0.5-1 μm[a]	10
Glass powder	3
Coarse glass fiber (code 110), median diameter = 1.8 μm	0

[a]From Wagner et al. (1973).

Source: Wagner et al. (1976).

TABLE 4-9. DOSE-RESPONSE DATA FOLLOWING INTRAPLEURAL ADMINISTRATION OF ASBESTOS TO RATS

Material	Dose mg	Number of rats with mesothelioma	Total number of rats	Percent of rats with tumors
SFA chrysotile	0.5	1	12	8
	1	3	11	27
	2	5	12	42
	4	4	12	33
	8	8	12	62
Crocidolite	0.5	1	11	9
	1	0	12	0
	2	3	12	25
	4	2	13	15
	8	5	11	45

Source: Wagner et al. (1973).

4.7 INTRATRACHEAL INJECTION

Intratracheal injection has been used to study the combined effect of the administration of chrysotile with benzo(a)pyrene in rats and hamsters. No lung tumors were observed in rats given 3 doses of 2 mg of chrysotile (Shabad et al., 1974) and in hamsters given 12 mg of chrysotile (Smith et al., 1970). However, co-administration of benzo(a)pyrene resulted in lung tumors, which suggests a co-carcinogenic or synergistic effect.

4.8 INTRAPERITONEAL ADMINISTRATION

Intraperitoneal injections of 20 mg of crocidolite or chrysotile produced 3 peritoneal mesotheliomas in 13 Charles River CD rats, but 20 mg of amosite produced no tumors in a group of 11 rats (Maltoni and Annoscia, 1974). Maltoni and Annoscia also injected 25 mg of crocidolite into 50 male and 50 female 17-week-old Sprague-Dawley rats and observed 31 mesothelial tumors in males and 34 in females.

In an extensive series of experiments, Pott and Friedrichs (1972) and Pott et al. (1976) produced peritoneal mesotheliomas in mice and rats that were injected with various commercial varieties of asbestos and other fibrous material. These results are shown in Table 4-10. Using experiments with intrapleural administration, the malignant response was altered by ball-milling the fibers for 4 hours. The rate of tumor production was reduced from 55 to 32 percent and the time from onset of exposure to the first tumor was lengthened from 323 to 400 days following administration of 4 doses of 25 mg of UICC Rhodesian chrysotile. In the case of the ball-milled fibers, 99 percent of the fibers were reported to be smaller than 3 μm, 93 percent were smaller than 1 μm, and 60 percent were smaller than 0.3 μm.

Pott (1980) proposed a model for the relative carcinogenicity of mineral fibers, according to their dimensionality, using the results of injection and implantation data. Figure 4-5 shows the schematic features of this model. The greatest carcinogenicity is attributed to fiber lengths between 5 and 40 μm with diameters between 0.05 and 1 μm.

A strong conclusion that can be drawn from the above experimental data is that long (>4 μm) and fine diameter (<1 μm) fibers are more carcinogenic than short, thick fibers when they are implanted on the pleura or injected into the peritoneum of animals. The origin of a reduction in carcinogenicity for

TABLE 4-10. TUMORS IN ABDOMEN AND/OR THORAX OF RATS AFTER INTRAPERITONEAL INJECTION OF GLASS FIBERS, CROCIDOLITE, OR CORUNDUM

Dust	Form[a]	Intraperitoneal dose mg	Effective number of dissected rats	Number of days before first tumor	Average survival time of rats with tumors, days after injection	Rats with tumors, percent	Tumor/type[b] 1	2	3	4	5	6
Glass fibers MN 104	f	2	73	421	703	27.4	17	3	-	-	1	1
Glass fibers MN 104	f	10	77	210	632	53.2	36	4	-	1	3	-
Glass fibers MN 104	f	2 x 25	77	194	367	71.4	47	6	2	-	-	-
Crocidolite	f	2	39	452	761	38.5	12	3	-	-	2	1
Corundum	g	2 x 25	37	545	799	8.1	1	-	-	2	2	2
UICC Rhodesian chrysotile	f	2	37	431	651	16.2	4	2	-	-	1	-
UICC Rhodesian chrysotile	f	6.25	35	343	501	77.1	24	3	-	-	-	-
UICC Rhodesian chrysotile	f	25	31	276	419	0.6	21	2	1	1	-	-
UICC Rhodesian chrysotile	f	4 x 25	33	323	361	54.5	16	2	-	-	-	-
UICC Rhodesian chrysotile	f	3 x 25 s.c.	33	449	449	3.0	-	-	1 s.c.	-	-	-
UICC Rhodesian milled	f	4 x 25	37	400	509	32.4	9	3	-	-	-	-
Palygoescite	f	3 x 25	34	257	348	76.5	24	2	-	-	-	-

TABLE 4-10. (continued)

Dust	Form[a]	Intraperitoneal dose mg	Effective number of dissected rats	Number of days before first tumor	Average survival time of rats with tumors, days after injection	Rats with tumors, percent	Tumor/type[b] 1	2	3	4	5	6
Glass fibers S + S 106	f	2	34	692	692	2.9	1	-	-	-	-	-
Glass fibers S + S 106	f	10	36	350	530	11.1	2	2	-	-	1	-
Glass fibers S + S 106	f	4 x 25	32	197	325	71.9	20	3	-	-	-	-
Gypsum	f	4 x 25	35	579	583	5.7	-	-	1	1	1	-
Henalite	f	4 x 25	34	249	315	73.5	17	8	-	-	-	-
Actinolite	g	4 x 25	39	-	-	-	-	-	-	-	-	-
Biotite	g	4 x 25	37	-	-	-	-	-	-	-	-	-
Haematite (precipitation)	g	4 x 25	34	-	-	-	-	-	-	-	-	-
Haematite (mineral)	g	4 x 25	38	-	-	-	-	-	-	-	-	-
Pectolite	g	4 x 25	40	569	569	2.5	-	-	-	1	1	1
Sanidine	g	4 x 25	39	579	579	2.6	-	1	-	-	-	-
Talc	g	4 x 25	36	587	587	2.8	1	-	-	-	-	-
NaCl (control)	-	4 x 2 ml	72	-	-	-	-	-	-	-	-	-

[a] f = fibrous; g = granular.

[b] Tumor Types are: 1 Mesothelioma; 2 Spindle cell sarcoma; 3 Polym-cell sarcoma; 4 Carcinoma; 5 Reticulum cell sarcoma; 6 Benign -- not evaluated in tumor rates.

Sources: Pott and Friedrichs (1972); Pott et al. (1976).

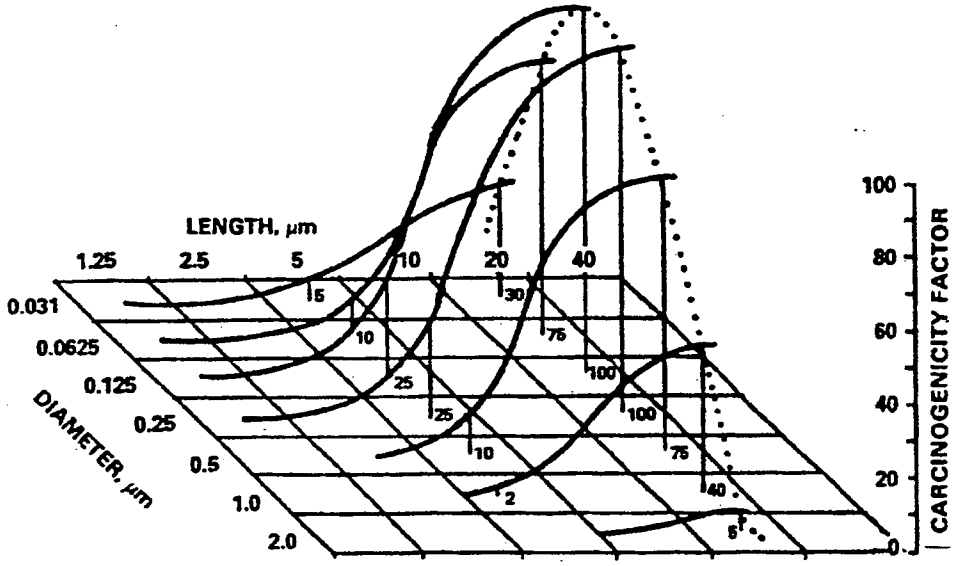

Figure 4-5. Hypothesis concerning the carcinogenic potency of a fiber as a function of its length and width using data on tumor incidence from injection and implantation studies.

Source: Pott (1980).

shorter, ball-milled fibers is less clear because the relative contributions of shorter fiber length and the significant alteration of the crystal structure by input of physical energy have not yet been defined. Extrapolation of data on size-dependent effects obtained from intrapleural or intraperitoneal administration, to inhalation, where movement of the fibers in airways and subsequently through body tissues is strongly size-dependent, presents significant difficulties. The number of shorter (<5 μm) fibers in an exposure circumstance may be 100 times greater than the number of longer fibers; therefore, their carcinogenicity must be 1/100 times as much before their contribution can be neglected.

4.9 TERATOGENICITY

There is no evidence that asbestos is teratogenic. Schneider and Maurer (1977) fed pregnant CD-1 mice doses of 4-400 mg/kg body weight (1.43 to 143) for gestation days 1 to 15. They also administered 1, 10, or 100 μg of asbestos to 4-day blastocysts, which were transferred to pseudopregnant mice. No positive effects were noted in either experiment.

4.10 SUMMARY

Animal data on the carcinogenicity of asbestos fibers confirm and extend epidemiological human data. Mesothelioma and lung cancer are produced by all the principal commercial asbestos varieties, chrysotile, amosite, crocidolite, and anthophyllite, even by exposures as short as one day. The deposition and clearance of fibers from the lung suggest that most inhaled fibers (~99 percent) are eventually cleared from the lung by ciliary or phagocytic action. Chrysotile appears to be more readily removed, and dissolution of the fibers occurs in addition to other clearance processes. Implantation and injection studies suggest that the carcinogenicity of durable mineral fibers is related to their dimensionality and not to their chemical composition. Long (≥4 μm) and thin (≤1 μm) fibers are most carcinogenic when they are in place at a potential tumor site. However, deposition, clearance, and migration of fibers are also size dependent, and the importance of all size-dependent effects in the carcinogenicity of inhaled fibers is not fully established.

5. ENVIRONMENTAL EXPOSURES TO ASBESTOS

5.1 INTRODUCTION

The analysis of ambient air samples for asbestos has utilized techniques different from those used in occupational circumstances. This situation occurred because typical urban air may contain up to 100 $\mu g/m^3$ of particulate matter in which the researcher is attempting to quantify asbestos concentrations from about 0.1 ng/m^3 to perhaps 1000 ng/m^3. Thus, asbestos may constitute only 0.0001 to 1 percent of the particulate matter in a given air sample. Asbestos found in ambient air has a size distribution such that the vast majority of fibers are too short or too thin to be seen with an optical microscope. In many cases, these fibers and fibrils will be agglomerated with a variety of other materials present in the air samples.

The only effective method of analysis uses electron microscopy to enumerate and size all asbestos fibers (Nicholson and Pundsack, 1973; Samudra et al., 1978). Samples for such analysis are usually collected either on a Nuclepore® (polycarbonate) filter with a pore size of 0.4 µm or on a Millipore® (cellulose ester) filter with a pore size of 0.8 µm. In some cases the Millipore® is backed by nylon mesh. Samples collected on Nuclepore® filters are prepared for direct analysis by carbon coating the filter to entrap the collected particles. A segment of the coated filter is then mounted on an electron microscope grid, which is placed on a filter paper saturated with chloroform so that the chloroform vapors dissolve the filter material. (Earlier electron microscopic analysis utilized a rub-out technique in which the ash residue was dispersed in a nitrocellulose film on a microscope slide and a portion of the film was then mounted on an electron microscope grid for scanning.)

Samples collected on Millipore® filters are prepared for indirect analysis by ashing a portion of the filter in a low temperature oxygen furnace. This removes the membrane filter material and all organic material collected in the sample. The residue is recovered in a liquid phase, dispersed by ultrasonification, and filtered on a Nuclepore® filter. The refiltered material is coated with carbon and mounted on a grid as above. The samples are then subjected to analysis. Chrysotile asbestos is identified on the basis of its morphology in the electron microscope and amphiboles are identified by their selected area electron diffraction patterns, supplemented by energy-dispersive X-ray analysis. Fiber concentrations in fibers per unit of volume (such as $fibers/cm^3$,

fibers/m^3, etc.) are calculated based on sample volume and filter area counted. In some cases, mass concentrations are reported using fiber volume and density relationships. However, mass concentrations may not be reliable if the sample contains fibrous forms, such as clusters, bundles, and matrices, where fiber volume is difficult to determine. These materials may constitute most of the asbestos mass in some samples, particularly those reflecting emission sources. Current fiber counting methods do not include those clumps. However, many of them are respirable and to the extent that they are broken apart in the lungs into individual fibers, they may add to the carcinogenic risk. On the other hand, methods which break up fibers generally disperse the clumps as well. In such analyses, the clumps would contribute to the mass.

In much of the earlier analyses of chrysotile concentrations in the United States the ashed material was either physically dispersed or disrupted by ultrasonification. Thus, no information was obtained on the size distribution of the fibers in the original aerosol. Air concentrations were given only in terms of total mass of asbestos present in a given air volume, usually in nanograms per cubic meter (ng/m^3). (See Section 5-9 for data on the interconvertability of optical fiber counts and electron microscopic mass determinations.) With the use of Nuclepore® filters and appropriate care in the collection of samples and their processing, information on the fiber size distribution can be obtained and concentrations of fibers of selected dimensions can be calculated. Samples collected on Millipore® filters can be ashed and the residue resuspended and filtered through Nuclepore® filters. However, some breakage of fibers during the process is likely. Direct processing of Millipore® filters for electron microscopic analysis has been reported by Burdett and Rood (1983) and is being tested by several laboratories. However, the utility and reliability of this technique is unknown at present.

Ideally, one would like a measure of exposure that would be proportional to the carcinogenic risk. Unfortunately, this is not possible because of our limited information on the carcinogenicity of fibers according to length and width and the lack of information on the deposition, clearance, and movement through the body of fibers of different sizes. Secondly, our epidemiological evidence of disease relates to fibers longer than 5 μm measured by optical microscopy. It should be recognized that electron microscopic fiber counts of fibers longer than 5 μm of length will differ considerably from optical microscopy counts of the same sample because of the presence of a large number of

fibers undetected by optical microscopy. Nevertheless, it would appear that the best measure of risk would be electron microscopic fiber counts of fibers greater than 5 μm in length and use of an empirically determined adjustment for the increased resolving power of the electron microscope when such measurements are used for risk assessment.

Two of the studies described below provide information on fiber as well as mass concentrations. However, in one case (Constant, 1983) the fiber concentrations were of fibers of all length, and thus are impossible to translate into optical microscopic counts (other than by mass). While the other studies are limited because of the absence of fiber concentrations, they are sufficient to indicate exposure circumstances of concern or that warrant further investigation. Further, using an empirical conversion factor (having a very large uncertainty), estimates of environmental exposures can be made in terms of optical fiber counts.

Unfortunately, few studies have been conducted which provide data relating asbestos fiber concentrations and health effects. While estimates of asbestos concentrations based on conversions from fiber-mass relationships have an associated uncertainty, they are the best data available for such assessments. Future studies will hopefully be designed to measure fiber number, size, and type for correlation with health effects.

An analysis of 25 samples collected in buildings having asbestos surfacing material (some buildings showing evidence of contamination) demonstrated the inadequacy of phase contrast optical microscopic techniques for the quantification of asbestos (Nicholson et al., 1975). Figure 5-1 shows the correlation of optical fiber counts determined using National Institute for Occupational Safety and Health (1972) prescribed techniques and asbestos mass measurements obtained on the same samples. In determining the fiber concentrations, all objects with an aspect ratio of three or greater were enumerated using phase-contrast microscopy. Petrographic techniques were not utilized to verify whether an object was an asbestos fiber. Figure 5-1 shows that the optical microscopic data do not reflect the mass concentrations of asbestos determined by electron microscopy, largely because of a considerable number of nonasbestos fibers that were in the ambient air and were counted in the optical microscopic analysis.

The available published asbestos exposure data are to a large extent episodic in nature. The studies were not designed to provide measures of ambient concentrations throughout the United States. The data presented here

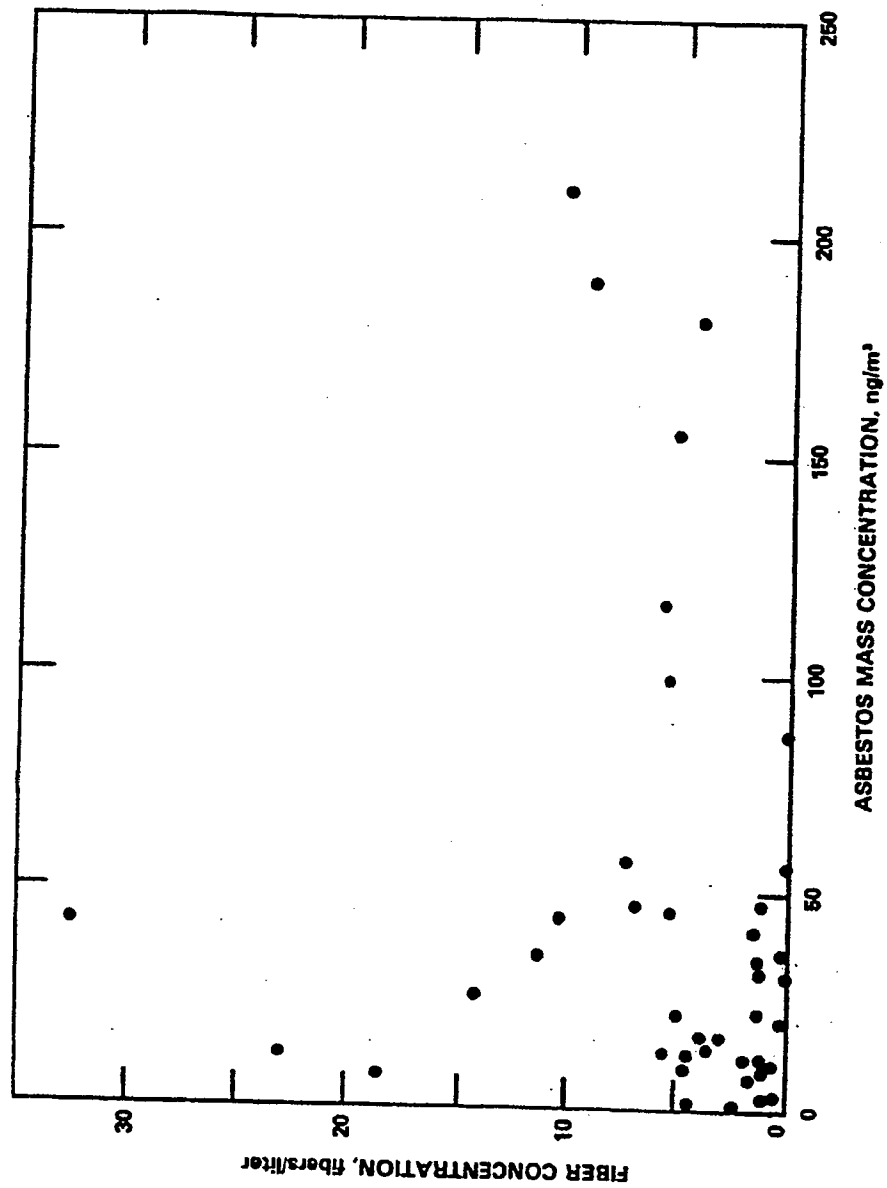

Figure 5-1. Fiber concentrations by optical microscopy versus asbestos mass concentrations by electron microscopy.

Source: National Institute for Occupational Safety and Health (1972).

represent the published data that are available. These data show what concentration can occur in the circumstances given. When useful information (i.e., number of sites, frequency of samples) is available that helps characterize the representativeness of exposure of the data, it is presented. But as can be seen, these data generally do not represent the results of systematic studies designed to characterize the ambient asbestos concentrations in the United States or those in typical building circumstances.

5.2 GENERAL ENVIRONMENT

Asbestos of the chrysotile variety has been found to be a ubiquitous contaminant of ambient air. A study of 187 quarterly samples collected in 48 U.S. cities in 1969-1970 showed chrysotile asbestos to be present in virtually all metropolitan areas (Nicholson, 1971; Nicholson and Pundsack, 1973). Table 5-1 lists the distribution of values obtained in that study, along with similar data obtained by the Battelle Memorial Institute (U.S. EPA, 1974). Each value represents the chrysotile concentration in a composite of from five to seven 24-hour samples, thus averaging possible peak concentrations which could occur periodically or randomly. Of the three samples greater than 20 ng/m^3 analyzed by Mount Sinai School of Medicine, one sample was in a city that had a major shipyard and another was in a city that had four brake manufacturing facilities with no emission controls. Thus, these samples may have included a contribution from a specific source in addition to that of the general ambient air. Also shown in Table 5-1 is the distribution of chrysotile concentrations from five-day samples of the air in Paris (Sebastien et al., 1980). These values were obtained during 1974 and 1975 and were generally lower than those measured in the United States, perhaps reflecting a diminished use of asbestos in construction compared to that of the United States during 1969-1970.

In a study of the ambient air of New York City, in which samples were taken only during daytime working hours, higher values than those mentioned above were obtained (Nicholson et al., 1971). These 4- to 8-hour samples were collected between 8:00 A.M. and 5:00 P.M., and they reflect what could be intermittently higher concentrations during those hours compared to nighttime periods. Table 5-2 records the chrysotile content of 22 samples collected in the five boroughs of New York and their overall cumulative distribution. The samples analyzed in all the studies discussed above were taken during a period when fireproofing of high rise buildings by spraying asbestos-containing

TABLE 5-1. CUMULATIVE DISTRIBUTION OF 24-HOUR CHRYSOTILE ASBESTOS CONCENTRATIONS IN THE AMBIENT AIR OF U.S. CITIES AND PARIS, FRANCE

	Electron Microscopy Analysis				
	Mount Sinai School of Medicine[a]		Battelle Memorial Institute[b]		Paris, France[c]
Concentration (ng/m^3) less than	Number of samples	Percentage of samples	Number of samples	Percentage of samples	Percentage of samples
1.0	61	32.6	27	21.3	70
2.0	119	63.6	60	47.2	85
5.0	164	87.7	102	80.1	98
10.0	176	94.2	124	97.6	100
20.0	184	98.5	125	98.5	
50.0	185	99.0	127	100.0	
100.0	187	100.0	127	100.0	

Sources: [a]Nicholson (1971); [b]U.S. EPA (1974); [c]Sebastien et al. (1980).

materials was permitted. The practice was especially common in New York City. While no sampling station was known to be located adjacent to an active construction site, unusually high levels could nevertheless have resulted from this procedure. Other sources that may have contributed to these air concentrations include automobile braking, other construction activities, consumer use of asbestos products, and maintenance or repair of asbestos-containing materials (e.g., thermal insulation).

5.3 CHRYSOTILE ASBESTOS CONCENTRATIONS NEAR CONSTRUCTION SITES

To determine if construction activities could be a significant source of chrysotile fiber in the ambient air, 6- to 8-hour daytime sampling was conducted in lower Manhattan in 1969 near sites where extensive spraying of asbestos-containing fireproofing material was taking place. Eight sampling sites were established near the World Trade Center construction site during the period when asbestos material was sprayed on the steelwork of the first tower.

TABLE 5-2. DISTRIBUTION OF 4- TO 8-HOUR DAYTIME CHRYSOTILE ASBESTOS CONCENTRATIONS IN THE AMBIENT AIR OF NEW YORK CITY, 1969-1970

Asbestos concentration (ng/m^3) less than	Cumulative number of samples	Cumulative percentage of samples
1	0	0.0
2	1	4.5
5	4	18.1
10	8	36.4
20	16	72.7
50	21	95.4
100	22	100.0

Distribution by borough

Sampling locations	Number of samples	Asbestos air level, ng/m^3	
		Range	Average
Manhattan	7	8-65	30
Brooklyn	3	6-39	19
Bronx	4	2-25	12
Queens	4	3-18	9
Staten Island	4	5-14	8

Source: Nicholson et al. (1971).

Table 5-3 shows the results of building-top air samples taken at sites within one-half mile of the Trade Center site, demonstrating that spray fireproofing did contribute significantly to asbestos air pollution (Nicholson et al., 1971; Nicholson and Pundsack, 1973). In some instances, chrysotile asbestos levels were observed that were approximately 100 times greater than the concentrations typically found in ambient air.

5.4 ASBESTOS CONCENTRATIONS IN BUILDINGS IN THE UNITED STATES AND FRANCE

During 1974, 116 samples of indoor and outdoor air were collected in 19 buildings (usually 4-6 indoor samples and 1 ambient air control sample per building) in 5 U.S. cities to assess whether contamination of the building air resulted from the presence of asbestos-containing surfacing materials in rooms or return air plenums (Nicholson et al., 1975). The asbestos materials in the buildings were of two main types: 1) a cementitious or plaster-like material that had been sprayed as a slurry onto steelwork or building surfaces, and

TABLE 5-3. DISTRIBUTION OF 6- TO 8-HOUR CHRYSOTILE ASBESTOS CONCENTRATIONS WITHIN ONE-HALF MILE OF THE SPRAYING OF ASBESTOS MATERIALS ON BUILDING STEELWORK, 1969-1970

Asbestos concentration (ng/m^3) less than	Cumulative number of samples	Cumulative percentage of samples
5	0	0.0
10	3	17.6
20	8	47.1
50	14	82.3
100	16	94.1
200	16	94.1
500	17	100.0

Distribution of chrysotile air levels according to distance from spray fireproofing sites

Sampling locations	Number of samples	Asbestos air level, ng/m^3	
		Range	Average
1/8-1/4 mile	11	9-375	60
1/4-1/2 mile	6	8-54	25
1/2-1 mile	5	3.5-36	18

Source: Nicholson et al. (1971).

2) a loosely bonded fibrous mat that had been applied by blowing a dry mixture of fibers and binders through a water spray onto the desired surface. The friability of the two types of materials differed considerably; the cementitious spray surfaces were relatively impervious to damage while the fibrous sprays were highly friable. The results of air sampling in these buildings (Table 5-4) provide evidence that the air of buildings with fibrous asbestos-containing materials may often be contaminated.

Similar data were obtained by Sebastien et al. (1980) in a survey of asbestos concentration in buildings in Paris, France. Sebastien surveyed 21 asbestos-insulated buildings; 12 had at least one measurement higher than 7 ng/m^3, the upper limit of the outdoor asbestos concentrations measured by these investigators. The distribution of 5-day asbestos concentrations in these buildings, along with 19 outdoor samples taken at the same time, is shown in Table 5-5. One particularly disturbing set of data by Sebastien et al. is the concentrations of asbestos measured after surfacing material was removed or repaired. The average of 22 such samples was 22.3 ng/m^3. However,

TABLE 5-4. CUMULATIVE DISTRIBUTION OF 8- TO 16-HOUR CHRYSOTILE ASBESTOS CONCENTRATIONS IN BUILDINGS WITH ASBESTOS-CONTAINING SURFACING MATERIALS IN ROOMS OR IN AIR PLENUMS

Asbestos concentration ng/m³ less than	Friable spray		Cementitious spray		Control samples	
	Number of samples	Percentage of samples	Number of samples	Percentage of samples	Number	Percentage
1	5	9.3	3	10.7	5	14.7
2	6	11.1	6	21.4	6	17.6
5	8	14.8	10	35.7	15	44.1
10	15	27.8	17	60.7	21	61.8
20	28	51.9	26	92.9	29	85.3
50	44	81.5	27	96.4	33	97.1
100	49	90.7	27	96.4	34	100.0
200	52	96.3	28	100.0		
500	53	98.1				
1000	54	100.0				
Arithmetic average concentration		48 ng/m³		14.5 ng/m³		12.7 ng/m³

Source: Nicholson et al. (1975; 1976).

TABLE 5-5. CUMULATIVE DISTRIBUTION OF 5-DAY ASBESTOS CONCENTRATIONS IN PARIS BUILDINGS WITH ASBESTOS-CONTAINING SURFACING MATERIALS

Asbestos concentration (ng/m^3) less than	Building samples		Outdoor control samples	
	Number	Percentage	Number	Percentage
		Chrysotile		
1	57	42.2	14	73.7
2	70	51.9	16	84.2
5	92	68.1	17	89.5
10	104	77.0	19	100.0
20	117	86.7		
50	128	94.8		
100	129	95.6		
200	130	96.3		
500	132	97.8		
1000	135	100.0		
Arithmetic average concentration		25 ng/m^3		1 ng/m^3
		Amphiboles[a]		
1	112	83.0	19	100.0
2	115	85.2		
5	122	90.4		
10	125	92.6		
20	129	95.6		
50	131	97.0		
100	132	97.8		
200	133	98.5		
500	135	100.0		
Arithmetic average concentration		10 ng/m^3		0.1 ng/m^3

[a]No value reported for 104 building samples. Some materials would have contained no amphibole asbestos.

Source: Sebastien et al. (1980).

in two highly contaminated areas, significant reductions were measured (500 to 750 ng/m^3 decreased to less than 1 ng/m^3). The importance of proper removal techniques and cleanup cannot be overemphasized.

Sebastien et al. (1982) also measured concentrations of indoor airborne asbestos up to 170 ng/m^3 in a building with weathered asbestos floor tiles. Asbestos flooring is used in a large number of buildings and is the third largest use of asbestos fibers.

5.5 ASBESTOS CONCENTRATIONS IN U.S. SCHOOL BUILDINGS

Of concern was the discovery of extensive asbestos use in public school buildings (Nicholson et al., 1978). Asbestos surfaces were found in more than 10 percent of pupil-use areas in New Jersey schools, with two-thirds of the surfaces showing some evidence of damage. Because these values appear to be typical of conditions in many other states, it was estimated that 2 to 6 million pupils and 100,000 to 300,000 teachers may be exposed to released asbestos fibers in schools across the nation. To obtain a measure of contamination for this use of asbestos, 10 schools were sampled in the urban centers of New York and New Jersey and in suburban areas of Massachusetts and New Jersey. Schools were selected for sampling because of visible damage, in some cases extensive.

Table 5-6 lists the distribution of chrysotile concentrations found in samples taken over 4 to 8 hours in these 10 schools (1-5 samples per school). Chrysotile asbestos concentrations ranged from 9 ng/m^3 to 1950 ng/m^3, with an average of 217 ng/m^3. Outside air samples at 3 of the schools varied from 3 ng/m^3 to 30 ng/m^3, with an average of 14 ng/m^3. In all samples but two (which measured 320 ng/m^3) no asbestos was visible on the floor of the sampled area, although surface damage was generally present near the area. The highest value (1950 ng/m^3) was in a sample that followed routine sweeping of a hallway in a school with water damage to the asbestos surface, although no visible asbestos was seen on the hallway floor. It is emphasized that the schools were selected in testing on the basis of the presence of visible damage. Although the results cannot be considered typical of all schools having asbestos surfaces, the results do illustrate the extent to which contamination can exist.

A recent study suggests that the above school samples may not be atypical (Constant et al., 1983). Concentrations similar to those indicated above were found in the analysis of samples collected during a 5-day period in 25

TABLE 5-6. DISTRIBUTION OF CHRYSOTILE ASBESTOS CONCENTRATIONS IN 4- to 8-HOUR SAMPLES TAKEN IN PUBLIC SCHOOLS WITH DAMAGED ASBESTOS SURFACES

Asbestos concentration (ng/m^3) less than	Number of samples	Percentage of samples
5	0	0.0
10	1	3.7
20	1	3.7
50	6	22.2
100	12	44.4
200	19	70.4
500	25	92.6
1000	26	96.3
2000	27	100.0

Source: Nicholson et al. (1978).

schools that had asbestos surfacing materials. The schools were in a single district and were selected by a random procedure, not because of the presence or absence of damaged material. A population-weighted arithmetic mean concentration of 179 ng/m^3 was measured in 54 samples collected in rooms or areas that had asbestos surfacing material. In contrast, a concentration of 6 ng/m^3 was measured in 31 samples of outdoor air taken at the same time. Of special concern are 31 samples collected in the schools that used asbestos, but taken in areas where asbestos was not used. These data showed an average concentration of 53 ng/m^3, indicating dispersal of asbestos from the source. The data are summarized in Table 5-7. As published fiber counts were fibers of all sizes, only the fiber mass data are listed in the table. Additionally, fiber clumps were noted in many samples, but were not included in the tabulated masses.

A study commissioned by the Ontario Royal Commission (1984) of asbestos concentrations in buildings with asbestos insulation indicates levels comparable to that of urban air. It is not clear whether "insulation" is thermal insulation or sprayed surfacing material. Average concentrations (3-5 samples per building) ranged from less than 1 to 11 ng/m^3. However, during very careful maintenance and inspection work, concentrations substantially in excess of background were observed.

Sawyer (1977, 1979) reviewed a variety of data on air concentrations, measured by optical microscopy, for circumstances where asbestos materials in schools and other buildings are disturbed by routine or abnormal activity.

TABLE 5-7. CUMULATIVE DISTRIBUTION OF 5-DAY CHRYSOTILE ASBESTOS CONCENTRATIONS IN 25 SCHOOLS HAVING ASBESTOS SURFACING MATERIALS, 1980-1981

Asbestos concentration (ng/m^3) less than	Rooms with asbestos		Rooms without asbestos		Outdoor controls	
	Number of samples	Percentage of samples	Number of samples	Percentage of samples	Number of samples	Percentage of samples
			Chrysotile			
1	5	9.2	6	19.4	17	54.8
2	6	11.1	7	22.6	22	71.0
5	7	13.0	11	35.5	27	87.1
10	14	25.9	12	38.7	28	90.3
20	19	35.2	15	48.4	30	96.8
50	26	48.1	21	67.7	31	100.0
100	39	72.2	24	87.1		
200	45	83.3	29	93.5		
500	52	96.3	31	100.0		
1000	54	100.0				
Population weighted mean concentration		179 ng/m^3		53 ng/m^3		6 ng/m^3
			Amphiboles			
1	44	81.5	21	67.7	26	83.9
2	45	83.3	22	71.0	29	93.5
5	49	90.7	26	83.9	31	100.0
10	50	92.6	27	87.1		
20	52	96.3	27	87.1		
50	52	96.3	29	93.5		
100	54	100.0	31	100.0		
500						
Arithmetic mean concentration		3.6 ng/m^3		8.3 ng/m^3		0.5 ng/m^3

Source: Constant et al. (1983).

These results, shown in Table 5-8, demonstrate that a wide variety of activities can lead to high asbestos concentrations during disturbance of asbestos surfacing material. Maintenance and renovation work, particularly if performed improperly, can lead to substantially elevated asbestos levels.

TABLE 5-8. AIRBORNE ASBESTOS IN BUILDINGS HAVING FRIABLE ASBESTOS MATERIALS

Classification	Main mode of contamination	Activity description	Mean count of fibers per cm^3	n	Range or SD
Quiet, non-specific, routine	Fallout reentrainment	None	0.0	32	0.0
		Dormitory	0.1	NA	0.0-0.8
		University, schools	0.1	47	0.1
		Offices	0.2	14	0.1-0.6
Maintenance	Contact	Relamping	1.4	2	0.1
		Plumbing	1.2	6	0.1-2.4
		Cable movement	0.9	4	0.2-3.2
Custodial	Mixed: contact reentrainment	Cleaning	15.5	3	6.7
		Dry sweeping	1.6	5	0.7
		Dry dusting	4.0	6	1.3
		Bystander	0.3	3	0.3
		Heavy dusting	2.8	8	1.6
Renovation	Mixed: contact reentrainment	Ceiling repair	17.7	3	8.2
		Track light	7.7	6	2.9
		Hanging light	1.1	5	0.8
		Partition	3.1	4	1.1
		Pipe lagging	4.1	8	1.8-5.8
Vandalism	Contact	Ceiling damage	12.8	5	8.0

Source: Sawyer (1979).

5.6 CHRYSOTILE CONCENTRATIONS IN THE HOMES OF WORKERS

The finding of asbestos disease in family contacts of individuals occupationally exposed to chrysotile fibers directs attention to air concentrations in the homes of such workers. Thirteen samples were collected in the homes of asbestos mine and mill employees and analyzed for chrysotile (Nicholson et al., 1980). The workers were employed at mine operations in California and Newfoundland. At the time of sampling (1973 and 1976) they did not have

access to shower facilities nor did they commonly change clothes before going home. Table 5-9 lists the concentration ranges of the home samples. Three samples taken in homes of non-miners in Newfoundland yielded concentrations of 32, 45, and 65 ng/m^3. In contrast, the concentrations in workers' homes were much higher, pointing to the need for appropriate shower and change facilities at asbestos workplaces. Because asbestos-generated cancers have been documented in family contacts of workers, concentrations such as those described in this document should be viewed with particular concern.

TABLE 5-9. DISTRIBUTION OF 4-HOUR CHRYSOTILE ASBESTOS CONCENTRATIONS IN THE AIR OF HOMES OF ASBESTOS MINE AND MILL EMPLOYEES

Asbestos concentration (ng/m^3) less than	Number of samples	Percentage of samples
50	0	0.0
100	4	30.8
200	8	61.5
500	10	76.9
1000	12	92.3
2000	12	92.3
5000	13	100.0

Source: Nicholson et al. (1980).

5.7 SUMMARY OF ENVIRONMENTAL SAMPLING

Table 5-10 summarizes those studies of the general ambient air or of specific pollution circumstances that have a sufficient number of samples for comparative analysis. The data are remarkably consistent. Average 24-hour samples of general ambient air indicate asbestos concentrations of 1 to 2 ng/m^3 (two U.S. samples that may have been affected by specific sources were not included). Short-term daytime samples are generally higher, reflecting the possible contributions of traffic, construction, and other human activities. In buildings having asbestos surfacing materials, average concentrations 100 times greater than ambient air are seen in some schools and concentrations 5-30 times greater than ambient air are seen in some other buildings.

Figure 5-2 shows the cumulative distributions, on a log-probability plot, of the urban, school, and building samples. The straight lines in the data of Nicholson are suggestive of homogeneous sampling circumstances, but this may be fortuitous. The sampling situation of Constant et al. appears not to be homogeneous.

TABLE 5-10. SUMMARY OF ENVIRONMENTAL ASBESTOS SAMPLING

Sample set	Collection period	Number of samples	Mean Concentration, ng/m^3
Quarterly composites of 5 to 7 24-hour U.S. samples (Nicholson, 1971; Nicholson and Pundsack, 1973)	1969-70	187	3.3 C[a]
Quarterly composite of 5 to 7 24-hour U.S. samples (U.S. EPA, 1974)	1969-70	127	3.4C
5-day samples of Paris, France (Sebastien et al., 1980)	1974-75	161	0.96 C
6- to 8-hour samples of New York City (Nicholson et al., 1971)	1969	22	16 C
5-day, 7-hour control samples for U.S. school study (Constant et al., 1983)	1980-81	31	6.5 (6C, 0.5A[b])
16-hour samples of 5 U.S. cites (U.S. EPA, 1974)	1974	34	13 C
New Jersey schools with damaged asbestos surfacing materials in pupil use areas (Nicholson et al., 1978)	1977	27	217 C
U.S. school rooms/areas with asbestos surfacing material (Constant, 1983)	1980-81	54	183 (179C, 4A)
U.S. school rooms/areas in building with asbestos surfacing material (Constant, 1983)	1980-81	31	61 (53C, 8A)
Buildings with asbestos materials in Paris, France (Sebastien et al., 1980)	1976-77	135	35 (25C, 10A)
U.S. buildings with friable asbestos in plenums or as surfacing materials (Nicholson et al., 1975; Nicholson et al., 1976)	1974	54	48 C
U.S. buildings with cementitious asbestos material in plenums or as surfacing materials (Nicholson et al., 1975, 1976)	1974	28	15 C
Ontario buildings with asbestos insulation (Ontario Royal Commission, 1984)	1982	63	2.1

[a]C = chrysotile. [b]A = amphibole.

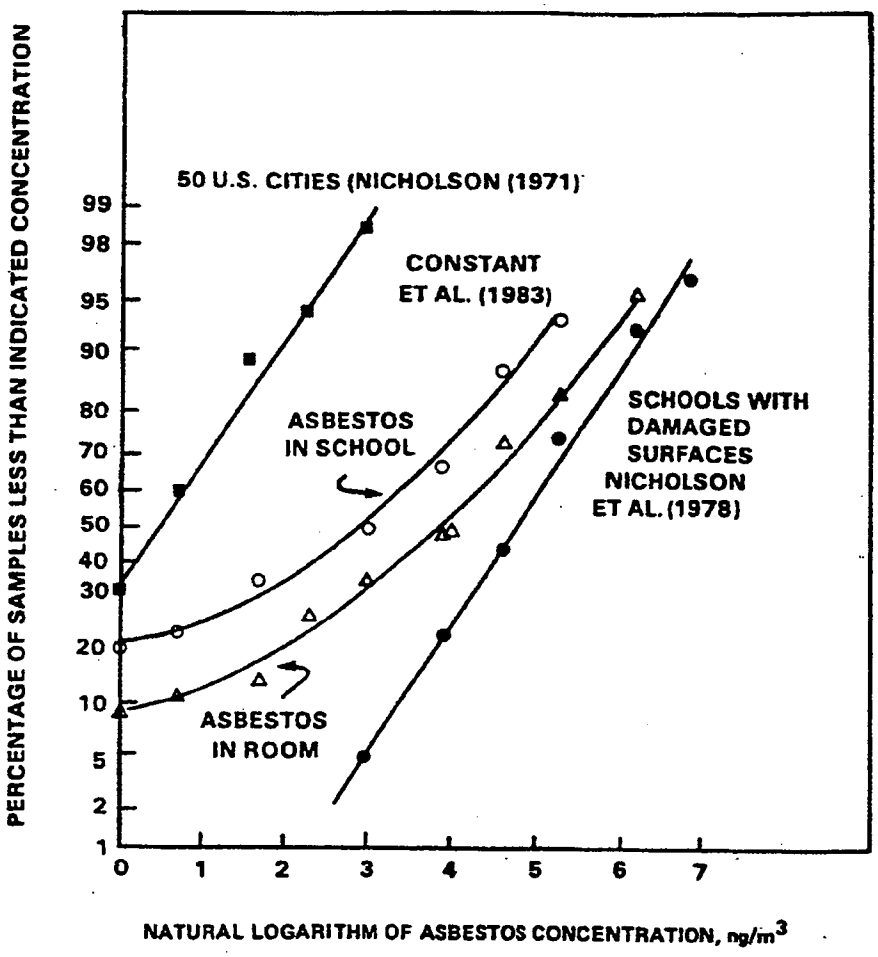

Figure 5-2. Cumulative distribution, on a log probability plot, of urban, school, and building asbestos air concentrations.

5.8 OTHER EMISSION SOURCES

Weathering of asbestos cement wall and roofing materials was shown to be a source of asbestos air pollution by analyzing air samples taken in buildings constructed of such material (Nicholson, 1978). Seven samples taken in a school after a heavy rainfall showed asbestos concentrations from 20-4500 ng/m^3 (arithmetic mean = 780 ng/m^3); all but two samples exceeded 100 ng/m^3. The source was attributed to asbestos washed from asbestos cement walkways and asbestos cement roof panels. No significantly elevated concentrations were observed in a concurrent study of houses constructed of asbestos cement materials. Roof water runoff from the homes landed on the ground and was not reentrained, while that of the schools fell to a smooth walkway, which allowed easy reentrainment when dry. Contamination from asbestos cement siding has also been documented by Spurny et al. (1980).

One of the more significant remaining contributions to environmental asbestos concentrations may be emissions from braking of automobiles and other vehicles. Measurements of brake and clutch emissions reveal that, annually, 2.5 tons of unaltered asbestos are released to the atmosphere and an additional 68 tons fall to roadways, where some of the asbestos is dispersed by passing traffic (Jacko et al., 1973).

5.9 INTERCONVERTIBILITY OF FIBER AND MASS CONCENTRATIONS

The limited data that relate asbestos disease to exposure are derived from studies of workers exposed in occupational environments. In these studies, concentrations of fibers longer than 5 μm were determined using optical microscopy or they were estimated from optical microscopy measurements of total particulate matter. All current measurements of low-level environmental pollution utilize electron microscopy techniques, which determine the total mass of asbestos present in a given volume of air. In order to extrapolate dose-response data obtained in studies of working groups to environmental exposures, it is necessary to establish a relationship between optical fiber counts and the mass of asbestos determined by electron microscopy.

Data are available relating optical fiber counts (longer than 5 μm) to the total mass of asbestos, as determined by electron microscopy techniques or other weight determinations. These relationships (Table 5-11) provide crude

TABLE 5-11. MEASURED RELATIONSHIPS BETWEEN OPTICAL FIBER COUNTS AND MASS AIRBORNE CHRYSOTILE

Sampling situation	Fiber[a] counts f/ml	Mass concentration µg/m³	Conversion factors µg/m³ / f/ml or µg / 10⁶f	10³ f/µg
Textile factory British Occupational Hygiene Society (1968) (weight vs. fiber count)	2	120	60	16
Air chamber monitoring Davis et al. (1978)	1950	10,000	5	200
Monitoring brake repair work Rohl et al. (1976) Electron Microscopy (E.M. mass vs. fiber count)	0.1 to 4.7 (7 samples)	0.1 to 6.6	0.7 to 24[b] mean = 6	170
Textile mill Lynch et al. (1970)			150[c]	6.7
Friction products manufacturing Lynch et al. (1970)			70[c]	13.9
Pipe manufacturing Lynch et al. (1970)			45[c]	22.5

[a]All fiber counts used phase-contrast microscopy and enumerated fibers longer than 5 µm.

[b]Conversion factor may be low due to losses in electron microscopy processing.

[c]Conversion factor may be high because of overestimate of asbestos mass on the basis of total magnesium.

estimates of a conversion factor relating fiber concentration in fibers per milliliter (f/ml) to airborne asbestos mass in micrograms per cubic meter ($\mu g/m^3$). The proposed standards for asbestos in Great Britain, set by the British Occupational Hygiene Society (BOHS), states that a "respirable" asbestos mass of 0.12 mg/m^3 is equivalent to 2 f/ml (British Occupational Hygiene Society, 1968). The standard does not state how this relationship was determined. If the relationship was obtained from magnesium determinations in an aerosol, the weight determination would likely be high because of the presence of other nonfibrous magnesium-containing compounds in the aerosol. Such was the case in the work of Lynch et al. (1970), and their values for the conversion factor are undoubtedly overestimates. The data of Rohl et al. (1976) are likely to be underestimates because of possible losses in the determination of mass by electron microscopy. No information exists on the procedures used to determine the mass of chrysotile in the data presented by Davis et al. (1978).

The range of 5 to 150 for the conversion factor relating mass concentration to optical fiber concentration is large and any average value derived from it has a large uncertainty. However, for the purpose of extrapolating to low mass concentrations from fiber count, the geometric mean of the above range of conversion factors, 30 $\mu g/m^3/f/ml$, will be used. The geometric standard deviation of this value is 4, and this uncertainty severely limits any extrapolation in which it is used. In the case of amosite, the data of Davis et al. (1978) suggest that a conversion factor of 18 is appropriate. However, these data yield lower chrysotile values than all other chrysotile estimates; therefore, they may also be low for amosite.

5.10 SUMMARY

Measurements using electron microscopy techniques established the presence of asbestos in the urban ambient air, usually at concentrations less than 10 ng/m^3. Concentrations of 100 ng/m^3 to 1000 ng/m^3 were measured near specific asbestos emission sources, in schools where asbestos-containing materials are used for sound control, and in office buildings where similar materials are used for fire control. Excess concentrations in buildings have usually been associated with visible damage or erosion of the asbestos materials. Many buildings with intact material have no increased concentrations of asbestos. Most ambient measurements were taken over ten years ago and it is very important to obtain more current data.

6. RISK EXTRAPOLATIONS AND HUMAN EFFECTS OF LOW EXPOSURES

6.1 RISK EXTRAPOLATIONS FOR LUNG CANCER AND MESOTHELIOMA

To obtain dose-response estimates at current or projected environmental asbestos concentrations, it is necessary to extrapolate from epidemiological data on deaths that have resulted from exposures to the considerably higher concentrations extant in occupational circumstances. As mentioned previously, the available data are compatible with a linear exposure-response relationship, with no evidence of a threshold. However, the limited data that indicate the validity of this relationship are for exposures two or three orders of magnitude higher than those of concern for environmental exposures.

The values determined for K_L and K_M in Chapter 3 are used to calculate best estimate risks from continuous exposures to 0.0001 and 0.01 f/ml. The values for continuous exposure were derived by multiplying 40 hr/wk risks, obtained from occupational exposures, by 4.2 (the ratio of hours in a week to 40 hours.) The lower concentration is typical of urban ambient air and corresponds to about 3 ng/m^3. The higher concentration, corresponding to about 300 ng/m^3, was measured in several environmental exposure circumstances. These two examples provide unit risks from which risk at other continuous exposures can be calculated as needed.

Tables 6-1, 6-2, and 6-3 list the calculated lifetime risks of mesothelioma and lung cancer for continuous exposures to 0.0001 and 0.01 f/ml of asbestos for various time periods. Risks from longer or shorter exposures can be estimated by directly scaling the data in the tables, as can risks from other concentrations (i.e., 0.1 f/ml). Equations 3-3a, 3-6c, 3-6d, and 3-6e and values of $K_L = 1.0 \times 10^{-2}$ and $K_M = 1.0 \times 10^{-8}$ were used in these calculations. The calculation uses a lifetable approach, in which the hypothetical population at risk is continuously decreased by its calculated mortality from all causes. Different overall mortality rates for smokers and non-smokers, as well as for males and females, lead to different estimated mesothelioma risks by smoking and gender, in Tables 6-1, 6-2, and 6-3. In the calculation of lung cancer risk it was assumed that the calculated asbestos-related risk continue following cessation of any hypothetical exposure. U.S. 1977 mortality rates (National Center for Health Statistics, 1977) are used as the basic data for the calculation. The tables utilize both smoking specific (Tables 6-1 and

TABLE 6-1. LIFETIME RISKS PER 100,000 FEMALES OF DEATH FROM MESOTHELIOMA AND LUNG CANCER FROM CONTINUOUS ASBESTOS EXPOSURES OF 0.0001 AND 0.01 f/ml ACCORDING TO AGE AT FIRST EXPOSURE, DURATION OF EXPOSURE, AND SMOKING[a]

Age at onset of exposure	Concentration = 0.0001 f/ml, years of exposure: 1	5	10	20	lifetime	Concentration = 0.01 f/ml, years of exposure: 1	5	10	20	lifetime
Mesothelioma in Female Smokers										
0	0.1	0.6	1.2	1.9	2.5	13.9	64.0	115.1	186.2	252.0
10	0.1	0.4	0.7	1.1	1.4	9.0	40.3	71.4	112.0	142.8
20	0.1	0.2	0.4	0.6	0.7	5.3	23.5	40.7	61.3	72.8
30	0.0	0.1	0.2	0.3	0.3	2.8	12.3	20.6	29.4	32.8
50	0.0	0.0	0.0	0.0	0.0	0.6	2.0	2.9	3.5	3.5
Lung Cancer in Female Smokers										
0	0.0	0.1	0.3	0.5	1.5	2.8	13.4	26.7	53.3	149.9
10	0.0	0.1	0.3	0.5	1.2	2.8	13.4	26.7	53.3	123.5
20	0.0	0.1	0.3	0.5	1.0	2.8	13.4	26.7	52.5	96.9
30	0.0	0.1	0.3	0.5	0.7	2.8	13.3	25.9	47.9	71.0
50	0.0	0.1	0.2	0.2	0.2	2.0	8.8	15.5	22.7	24.4
Mesothelioma in Female Nonsmokers										
0	0.1	0.7	1.2	2.0	2.7	14.8	68.2	122.8	199.4	272.2
10	0.1	0.4	0.8	1.2	1.6	9.5	43.4	81.2	121.2	155.8
20	0.1	0.3	0.4	0.7	0.8	5.7	25.6	44.4	67.2	80.6
30	0.0	0.1	0.2	0.3	0.4	3.1	13.6	23.0	32.9	36.8
50	0.0	0.0	0.0	0.0	0.0	0.6	2.2	3.4	4.1	4.1
Lung Cancer in Female Nonsmokers										
0	0.0	0.0	0.0	0.1	0.2	0.3	1.3	2.7	5.2	16.4
10	0.0	0.0	0.0	0.1	0.1	0.3	1.3	2.7	5.3	13.9
20	0.0	0.0	0.0	0.1	0.1	0.3	1.3	2.7	5.2	11.3
30	0.0	0.0	0.0	0.1	0.1	0.3	1.3	2.7	5.0	8.7
50	0.0	0.0	0.0	0.0	0.0	0.3	1.1	2.1	3.5	3.9

[a]The 95% confidence limit on the risk values for lung cancer for an unstudied exposure circumstance is a factor of 10. The 95% confidence limit on the risk values for lung cancer on the average determined from 11 unit exposure risk studies is a factor of 2.5. The 95% confidence limit on the risk values for mesothelioma for an unstudied exposure circumstance is a factor of 20. The 95% confidence limit on the risk values for mesothelioma for a studied circumstance can be reasonably averaged as a factor of 5. The values for continuous exposure were derived by multiplying 40 hr/wk risks, obtained from occupational exposures, by 4.2 (the ratio of hours in a week to 40 hours.)

TABLE 6-2. LIFETIME RISKS PER 100,000 MALES OF DEATH FROM MESOTHELIOMA AND LUNG CANCER FROM CONTINUOUS ASBESTOS EXPOSURES OF 0.0001 AND 0.01 f/ml ACCORDING TO AGE AT FIRST EXPOSURE, DURATION OF EXPOSURE, AND SMOKING[a]

Age at onset of exposure	Concentration = 0.0001 f/ml, years of exposure: 1	5	10	20	lifetime	Concentration = 0.01 f/ml, years of exposure: 1	5	10	20	lifetime
Mesothelioma in Male Smokers										
0	0.1	0.5	0.9	1.4	1.8	10.6	48.3	85.5	137.5	181.0
10	0.1	0.3	0.5	0.8	1.0	6.6	29.4	51.5	77.8	98.3
20	0.0	0.2	0.3	0.4	0.5	3.6	16.4	28.0	41.2	47.9
30	0.0	0.1	0.1	0.1	0.2	2.0	8.1	13.4	18.5	20.2
50	0.0	0.0	0.0	0.0	0.0	0.3	1.1	1.5	1.8	1.8
Lung Cancer in Male Smokers										
0	0.0	0.2	0.4	0.8	2.4	4.2	20.9	41.9	83.4	238.1
10	0.0	0.2	0.4	0.8	2.0	4.2	21.0	42.0	83.9	197.8
20	0.0	0.2	0.4	0.8	1.6	4.2	21.3	42.3	83.4	157.5
30	0.0	0.2	0.4	0.8	1.2	4.2	21.3	42.0	79.2	117.6
50	0.0	0.2	0.3	0.4	0.4	3.6	16.2	28.4	40.3	42.0
Mesothelioma in Male Nonsmokers										
0	0.1	0.6	1.0	1.6	2.2	12.5	57.0	102.3	164.5	220.1
10	0.1	0.4	0.6	1.0	1.2	7.8	35.3	62.6	97.3	122.6
20	0.0	0.2	0.4	0.5	0.6	4.5	20.4	35.1	52.4	61.7
30	0.0	0.1	0.2	0.2	0.3	2.4	10.5	17.5	24.6	26.9
50	0.0	0.0	0.0	0.0	0.0	0.4	1.5	2.2	2.7	2.7
Lung Cancer in Male Nonsmokers										
0	0.0	0.0	0.0	0.0	0.2	0.3	1.5	2.9	5.9	18.5
10	0.0	0.0	0.0	0.1	0.2	0.3	1.5	2.9	5.9	15.5
20	0.0	0.0	0.0	0.1	0.1	0.3	1.5	2.9	5.9	12.6
30	0.0	0.0	0.0	0.1	0.1	0.3	1.5	2.9	5.7	9.7
50	0.0	0.0	0.0	0.0	0.0	0.3	1.3	2.2	3.9	4.2

[a]The 95% confidence limit on the risk values for lung cancer for an unstudied exposure circumstance is a factor of 10. The 95% confidence limit on the risk values for lung cancer on the average determined from 11 unit exposure risk studies is a factor of 2.5. The 95% confidence limit on the risk values for mesothelioma for an unstudied exposure circumstance is a factor of 20. The 95% confidence limit on the risk values for mesothelioma for a studied circumstance can be reasonably averaged as a factor of 5. The values for continuous exposure were derived by multiplying 40 hr/wk risks, obtained from occupational exposures, by 4.2 (the ratio of hours in a week to 40 hours.)

TABLE 6-3. LIFETIME RISKS PER 100,000 PERSONS OF DEATH FROM MESOTHELIOMA AND LUNG CANCER FROM CONTINUOUS ASBESTOS EXPOSURES OF 0.0001 AND 0.01 f/ml ACCORDING TO AGE AND DURATION OF EXPOSURE. U.S. GENERAL POPULATION DEATH RATES WERE USED AND SMOKING HABITS WERE NOT CONSIDERED[a]

Age at onset of exposure	Concentration = 0.0001 f/ml, years of exposure: 1	5	10	20	lifetime	Concentration = 0.01 f/ml, years of exposure: 1	5	10	20	lifetime
Mesothelioma in Females										
0	0.1	0.7	1.2	2.0	2.8	14.6	67.1	120.8	196.0	275.2
10	0.1	0.4	0.8	1.2	1.5	9.4	42.6	75.5	118.7	152.5
20	0.1	0.3	0.4	0.7	0.8	5.6	25.1	43.5	65.7	78.8
30	0.0	0.1	0.2	0.3	0.4	3.1	13.3	22.4	31.9	35.7
50	0.0	0.0	0.0	0.0	0.0	0.6	2.1	3.2	3.9	3.9
Lung Cancer in Females										
0	0.0	0.0	0.1	0.2	0.5	1.0	4.6	9.2	18.5	52.5
10	0.0	0.0	0.1	0.2	0.4	1.0	4.6	9.2	18.6	43.4
20	0.0	0.0	0.1	0.2	0.3	1.0	4.6	9.2	18.2	34.3
30	0.0	0.0	0.1	0.2	0.3	1.0	4.6	9.0	16.7	25.1
50	0.0	0.0	0.1	0.1	0.1	0.7	3.1	5.5	8.1	8.8
Mesothelioma in Males										
0	0.1	0.5	0.9	1.5	1.9	11.2	51.0	91.1	145.7	192.8
10	0.1	0.3	0.6	0.8	1.1	7.0	31.2	58.2	84.7	106.8
20	0.0	0.2	0.3	0.4	0.5	4.1	17.5	30.1	44.5	51.7
30	0.0	0.1	0.1	0.2	0.2	2.1	8.8	14.6	20.4	22.3
50	0.0	0.0	0.0	0.0	0.0	0.3	1.1	1.8	2.0	2.1
Lung Cancer in Males										
0	0.0	0.1	0.3	0.6	1.7	2.9	14.8	29.7	59.2	170.5
10	0.0	0.1	0.3	0.6	1.4	2.9	14.9	29.8	59.5	142.0
20	0.0	0.2	0.3	0.6	1.1	3.1	15.0	30.0	59.4	113.0
30	0.0	0.1	0.3	0.6	0.8	3.1	14.9	29.8	56.6	84.8
50	0.0	0.1	0.2	0.3	0.3	2.5	11.5	20.3	29.1	30.2

[a]The 95% confidence limit on the risk values for lung cancer for an unstudied exposure circumstance is a factor of 10. The 95% confidence limit on the risk values for lung cancer on the average determined from 11 unit exposure risk studies is a factor of 2.5. The 95% confidence limit on the risk values for mesothelioma for an unstudied exposure circumstance is a factor of 20. The 95% confidence limit on the risk values for mesothelioma for a studied circumstance can be reasonably averaged as a factor of 5. The values for continuous exposure were derived by multiplying 40 hr/wk risks, obtained from occupational exposures, by 4.2 (the ratio of hours in a week to 40 hours.)

6-2) and general population (Table 6-3) rates. We are assuming that the current U.S. male mortality rates reflect the experience of 67 percent smokers (many, however, are now ex-smokers) and that current female mortality rates reflect the experience of 33 percent smokers. Using these percentages and the data of Hammond (1966) on the mortality ratio of smokers to nonsmokers, smoking-specific total mortality rates are calculated. Current lung cancer mortality rates for males are multiplied by 1.5 to represent the rates for smoking males. The multiplication factor comes from the fact that the current male rates result from a population where 67 percent of men are smokers or ex-smokers. Correspondingly, current female lung cancer mortality rates are multiplied by 3 to reflect the fact that approximately 33 percent of women are current or ex-smokers. This factor for women may be low, because the current rapid increase in female rates may not yet fully reflect the full impact of women's smoking; however, they should not exceed the male smoker's rates. Nonsmoking lung cancer rates for both males and females are taken from Garfinkel (1981).

The results show the importance of the time course of mesothelioma. Children exposed at younger ages are especially susceptible because of their long life expectancy. The time of exposure plays little role in the lifetime excess risk of lung cancer; any exposure before the age of 45 or 50 contributes equally to the lifetime risk. The risk estimates are uncertain because of the variability of the data from which values of K_L are calculated and from uncertainties in extrapolating from risks estimated at high occupational exposures to concentrations 1/100 and less. Thus, actual risks in a given environmental exposure could be outside the listed ranges.

The risks in tables 6-1, 6-2, and 6-3 would appear to be the best estimates for exposure to fibers released from the variety of asbestos products used in the United States, including products containing small amounts of crocidolite and substantial quantities of amosite. As noted in the tables, the 95 percent confidence limits on the risk estimate for an unstudied exposure circumstance are a factor of times 1/10 and times 10. As indicated in section 3.17, exposures to crocidolite appear to carry a proportionately greater mesothelioma risk. Thus tables 6-1, 6-2, and 6-3 will likely underestimate (by perhaps a factor of 4) the mesothelioma risk to aerosols containing predominantly crocidolite asbestos. Conversely, in some pure chrysotile exposure circumstances (such as in mining and milling), the risk will be overestimated.

6.1.1 Alternative Analyses

As discussed previously, the data strongly support a relative risk model for lung cancer and a linear dose-response relationship. No data indicate the existence of a threshold, although one cannot be ruled out.

If a threshold does exist, there would be a corresponding reduction in the calculated lung cancer risk. There is no evidence of a quadratic term in the dose-response relationship nor is it indicated by existing models for asbestos lung cancer. If, however, a small quadratic term is present, there would be some reduction in the calculated risk.

Alternative models do exist for mesothelioma. There are uncertainties in the power of time at which mesothelioma risk increases. The uncertainty, however, has relatively little effect on calculated lifetime risk values, because a fit must be made to existing occupational risk over a time span of four or five decades, leaving only two or three decades of life for manifestation of different power function effects. A lower power requires a much greater multiplying coefficient. Table 6-4 shows the effect on the calculated lifetime risk of three different time functions that are matched to best fit the time course of risk among insulation workers. Table 6-4 shows that the extremes of effect differ by less than a factor of two. As was shown in Table 3-4, there is very little empirical evidence for quadratic or higher terms in the mesothelioma dose-response relationship, although they are compatible with existing cancer models. If higher than linear terms were present, they would reduce the calculated risks by less than a factor of two.

TABLE 6-4. COMPARISON OF THE EFFECT OF DIFFERENT MODELS FOR THE TIME COURSE OF MESOTHELIOMA RISK FOR A FIVE-YEAR EXPOSURE TO 0.01 F/ML

Age at onset of exposure	Calculated deaths/100,000 males		
	Eq. 3-6	t^5	$t^{3.2}$
0	51.0	76.0	46.0
10	31.2	38.0	27.2
20	17.5	17.5	15.0
30	8.8	7.0	7.0
50	1.1	1.0	1.0

6.2 OBSERVED ENVIRONMENTAL ASBESTOS DISEASE

Asbestos-related disease in persons who have not been directly exposed at the workplace has been reported since 1960. In that year, Wagner et al. (1960) published a review of 47 cases of mesothelioma found in the Northwest Cape Province of South Africa in the previous 5 years. Approximately half of the cases described were in individuals who, decades before, had lived or worked near an area of asbestos mining. The hazard from environmental asbestos exposure was further documented in the findings of Newhouse and Thomson (1965), showing that mesothelioma could occur among individuals whose potential asbestos exposure consisted of having resided near an asbestos factory or in the household of an asbestos worker; 20 of 76 cases from the files of the London Hospital were the result of such exposures.

Of considerable importance are data on the prevalence of X-ray abnormalities and the incidence of mesothelioma in family contacts of amosite factory employees in Paterson, New Jersey. Anderson and Selikoff (1979) showed that 35 percent of 685 family contacts of former asbestos factory workers had abnormalities characteristic of asbestos exposure when they were X-rayed 30 or so years after their first household contact. The data, shown in Tables 6-5 and 6-6, compare the household group with 326 New Jersey urban residents. The overall difference in the percentage of abnormalities between the two groups is highly significant. Of special concern is the finding that the difference in the prevalence of abnormalities in a group of children born into a worker's household after his employment ceased is also significant.

Four mesothelioma cases also occurred among the family contacts of these same factory workers (Anderson et al., 1976). Table 6-7 lists the cases by time from onset of exposure, along with the number of deaths from other causes in the same time period (1961-1977; one death occurred subsequent to 1977). One percent of the deaths after 20 years from first exposure were from mesothelioma; however, further observations will be necessary to fully establish the incidence of this neoplasm among family contacts. An additional contribution of asbestos-related lung cancer could also exist, but studies in this regard have not yet been completed.

A second population-based mortality study of mesothelioma and other cancer risks in environmental circumstances is that of Hammond et al. (1979b). This study compared the mortality of a group of 1779 residents within 0.5 mile of the Paterson amosite asbestos plant with 3771 controls in a different, but

TABLE 6-5. PREVALENCE OF RADIOGRAPHIC ABNORMALITIES ASSOCIATED WITH ASBESTOS EXPOSURE AMONG HOUSEHOLD MEMBERS OF AMOSITE ASBESTOS WORKERS

Exposure group	Total examined	One or more radiographic abnormalities present*	
New Jersey urban residents**	326	15 (5%)	
Entered household after active worker employment ceased†	40	6 (15%)	$\chi^2 = 7.1$ p <.01
Household resident during active worker employment†	685	240 (35%)	$\chi^2 = 114$ p <.001
Household resident and personal occupational asbestos exposure	51	23 (45%)	

*ILO U/C Pneumoconiosis Classification categories; irregular opacities 1/0 or greater; pleural thickening; pleural calcification; pleural plaques.

**No known direct occupational or household exposure to asbestos.

†No known direct occupational exposure to asbestos.

Source: Anderson and Selikoff (1979).

TABLE 6-6. CHEST X-RAY ABNORMALITIES AMONG 685 HOUSEHOLD CONTACTS OF AMOSITE ASBESTOS WORKERS AND 326 INDIVIDUAL RESIDENTS IN URBAN NEW JERSEY, A MATCHED COMPARISON GROUP

Group	Total examined	Pleural thickening present	Pleural calcification present	Pleural plaques present	Irregular* opacities present
Household contacts of asbestos workers	685	146 (18.8%)	66 (8.5%)	61 (7.9%)	114 (16.6%)
Urban New Jersey residents	326	4 (1.2%)	0 (0.0%)	2 (0.6%)	11 (3.4%)

*ILO U/C Pneumoconiosis Classification irregular opacities 1/0 or greater.

Source: Anderson and Selikoff (1979).

TABLE 6-7. MESOTHELIOMA FOLLOWING ONSET OF FACTORY ASBESTOS EXPOSURE, 1941-1945[a]

Years from onset	Factory workers (933)		Household contacts (2205)	
	Total deaths	Mesothelioma	Total deaths	Mesothelioma
<20 years	270	0	280	0
20-24 years	102	2	93	0
25-29 years	113	5	111	0
30-34 years	84	7	124	3
35+ years	5	0	56	1
Total >20 years	304	14	384	4
Total all years	574	14	664	4

[a]Data of Selikoff and Anderson.

Source: Nicholson (1981).

economically similar section of town. No differences in the relative mortality experiences are seen, except for one mesothelioma in the neighborhood group. This one case was an electrician; thus, occupational exposure may have contributed to the disease.

6.3 COMPARISON OF OBSERVED MORTALITY WITH EXTRAPOLATED DATA

The mortality data in these two population-based studies can be compared with estimates from the data that led to Table 6-3 but calculated for 35 years, rather than a lifetime. If the air concentration in both circumstances was 200 ng/m^3, approximately 2 mesothelioma deaths/100,000 would be expected in 35 years of observation. In both cases, the exposed population was about 2000; so, the expected number of mesotheliomas would be 0.04 (range: 0.004 to 0.4). The higher numbers observed, particularly in the household group, suggest that higher exposures (e.g., from shaking dusty overalls) may have occurred in workers' homes or that the extrapolations based on occupational data may understate risks.

6.4 COMPARISON OF ESTIMATED MESOTHELIOMAS WITH SEER DATA

The risk estimates of Table 6-1 through 6-3 can also be used to compare estimated mesothelioma risk with that observed in the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) Cancer Registry Program. Between 1973 and 1978, 170 cases of mesothelioma were identified among females in the SEER program which is based on 10% of the U.S. population (Connelly, 1980). Thus, about 280 cases occur annually in the U.S. among females. Using Equations 3-6d and the current female population of the U.S., it is estimated that 32 cases would occur annually from a continuous lifetime exposure to 0.0001 f/ml (about 3 ng/m^3). However, such a concentration, which was measured in urban areas during 1970-71 would be influenced by the substantial use of asbestos building products. The "background" concentrations during 1910-1940 would likely be less. Nicholson (1983) has estimated that about 20 mesotheliomas would occur among men and women if an average concentration of 2 ng/m^3 existed from 1930.

6.5 LIMITATIONS TO EXTRAPOLATIONS AND ESTIMATIONS

The above calculations of unit risk values for asbestos must be viewed with caution because they are uncertain and are necessarily based on estimates that are subjective, to some extent, because of the following limitations in data: (1) extrapolation from high occupational levels to much lower ambient levels, (2) mass-to-fiber conversion is uncertain, (3) various confounding aspects of the medical data and, very importantly (4) the nonrepresentative nature of the exposure estimates. The ranges of uncertainty estimated may in fact be greater than those stated here, but insufficient information exists by which to make more precise or definite estimates of uncertainty.

7. OTHER REVIEWS OF ASBESTOS HEALTH EFFECTS

7.1 INTRODUCTION

Recently several government agencies in different countries reviewed asbestos health effects. The most important of the reviews outside the United States are those of the Advisory Committee on Asbestos (1979a,b) (ACA) of the British Health and Safety Commission and the report of the Ontario Royal Commission (ORC) (1984). Updates on the British report have been published by Acheson and Gardner (1983), and most recently by Doll and Peto (1985). Each of these major reports was the result of lengthy testimony by many scientists and deliberation by a selected committee over a long period of time. In the United States, the National Academy of Sciences (NAS) has reviewed the non-occupational health risk of asbestiform fibers (National Academy of Sciences, 1984) and a Chronic Hazard Advisory Panel convened by the U.S. Consumer Product Safety Commission (1983) reported on the hazards of asbestos. There are large areas of agreement and some of disagreement between these other reviews and those of this document with regard to the spectrum of asbestos-related disease, the models describing asbestos-related lung cancer and mesothelioma, unit exposure risks in occupational circumstances, possible differences in carcinogenic potency of different asbestos minerals, and risk estimates at low, non-occupational exposures. These are discussed below.

7.2 THE SPECTRUM OF ASBESTOS-RELATED MORTALITY AND FIBER TYPE EFFECTS

There was unanimity that all commercial varieties of asbestos, including chrysotile, crocidolite, amosite, and anthophyllite, produced lung cancer in humans. The Ontario Royal Commission (1984) noted the considerable difference in lung cancer risk in different chrysotile-using processes. The reports implicated chrysotile, crocidolite and amosite in increased risks of mesothelioma. However, they disagreed on the importance of the role of each fiber type. The various British and Canadian reports view chrysotile as being a substantially less potent mesothelial carcinogen than amosite and amosite to be somewhat less potent than crocidolite. In the view of Acheson and Gardner (1983) "exposure to chrysotile alone so far has rarely been shown to cause mesothelioma." The British and Canadian views are based on the high frequency of mesothelioma deaths associated with crocidolite and amosite exposures, even

though, in some circumstances, the amphibole usage may have been very small relative to chrysotile. The CPSC report viewed chrysotile as being important in the production of pleural mesothelioma but not for peritoneal tumors. This view is based on similar ratios of pleural mesothelioma to excess lung cancer found among chrysotile-exposed workers compared to mixed or amphibole-exposed workers. The NAS believed that information was insufficient to establish a differential risk based on chemistry. It stated, "many of the apparent differences (in carcinogenic potency) may be explained by the differences in physical properties and concentrations used by the various industries."

All reports noted that the strength of the evidence associating asbestos exposure with cancers other than mesothelioma or of the lung is less. Gastrointestinal and laryngeal cancers were attributed to asbestos exposure by the Ontario Royal Commission (1984) and by the Advisory Committee on Asbestos (1979a,b), although Acheson and Gardner felt in 1983 that the evidence linking asbestos and GI cancer was "less convincing than in 1979." Doll and Peto (1985), in their review, conclude that there are no grounds for believing that gastrointestinal cancers in general are peculiarly likely to be caused by asbestos exposure. They further state that: (1) for laryngeal cancer, on the other hand, the evidence is quite strong; (2) they reserve judgment about the possibility that asbestos causes cancer of the esophagus; and (3) they also note what evidence would be needed to weaken their view regarding possible gastrointestinal tract cancer linkage to asbestos exposure. Both the U.S. Consumer Product Safety Commission Panel (1983) and National Academy of Sciences (1984) noted the increased risk of GI cancers in several cohorts, but each declined to take a firm position on causality. The CPSC Report specifically noted a disagreement on the issue among panelists.

7.3 MODELS FOR LUNG CANCER AND MESOTHELIOMA

All reports adopted models for lung cancer and mesothelioma similar to those of this report, a relative risk model for lung cancer and an absolute risk model for mesothelioma, in which the risk increased as a power function of time from exposure. All noted the limitations on the data establishing a dose-response relationship, but all felt a linear model was most appropriate, particularly for regulatory purposes. None suggested there was any evidence

of a threshold for asbestos cancer (although the data were insufficient to exclude one).

7.4 EXTRAPOLATIONS TO LOW EXPOSURE CIRCUMSTANCES

All of the major reviews by government agencies mentioned above undertook quantitative risk assessments for non-occupational or low exposures to asbestos. Because of agreement on the models for lung cancer and mesothelioma, very similar unit risks were estimated. Differences were largely the result of the choice of studies considered and were relatively small. All of the groups recognized the limitations in the data on which extrapolations were based, the dependence of the extrapolation on a linear dose-response relationship, the uncertainties of estimation of asbestos exposure in past years, and the difficulties of converting between different methods of measurement. Two groups (National Academy of Sciences, 1984; U.S. Consumer Product Safety Commission, 1983), estimated risks at lower exposures using average unit exposure risks as was done in this document; the other two (Ontario Royal Commission, 1984; Advisory Committee on Asbestos, 1979a,b) used risk estimates from data in different occupational studies and a range of the results was presented. Various estimates of the uncertainty of these risks were provided; most were of an ad hoc nature. A comparison of these different risk estimates is shown in Table 7-1. There is reasonable agreement between the estimates when consideration is taken of the different exposure circumstances. The NAS value for mesothelioma risk appears to be low relative to their lung cancer risk (the lifetime exposure risk barely exceeds that for lung cancer in a non-smoker). This may be the result of separately choosing b and k in the risk relationship $= bt^k$, rather than determining b after selecting a value for k.

When making the extrapolation from the work place exposure to the ambient exposure, one must be aware that the physical structure and other properties of asbestos may make the exposure risks substantially different.

TABLE 7-1. THE RISKS OF DEATH/100,000 INDIVIDUALS FROM MESOTHELIOMA AND LUNG CANCER FROM A LIFETIME ASBESTOS EXPOSURE TO 0.01 f/ml

Population	Lung cancer	Mesothelioma
This Document		
Female smokers	150.0 (15 - 1500)	252.0 (12.6 - 5040)
Female nonsmokers	16.4 (1.64 - 164)	272.0 (13.6 - 5440)
Male smokers	238.0 (23.8 - 2380)	181.0 (9.1 - 3620)
Male nonsmokers	18.5 (1.85 - 185)	220.0 (11.0 - 4400)
Males exposed 40 years from age 20 from Table 6-3	88.5 (8.9 - 885)	46.5 (2.3 - 920)
National Academy of Science (1984)		
Female smokers	57.5 (0 - 275)	22.5 (0 - 875)
Female nonsmokers	7.5 (0 - 32.5)	22.5 (0 - 875)
Male smokers	160.0 (0 - 725)	22.5 (0 - 875)
Male nonsmokers	15.0 (0 - 55)	22.5 (0 - 875)
U.S. Consumer Product Safety Commission (1983)		
Female smokers	95.2 (30.1 - 301.2)	246.0 (78.0 - 779.9)
Female nonsmokers	15.7 (5.0 - 496)	266.6 (84.3 - 842.9)
Male smokers	155.0 (49.0 - 490.1)	174.2 (55.1 - 551.0)
Male nonsmokers	17.5 (5.54 - 55.4)	215.3 (68.1 - 680.8)
Ontario Royal Commission[a] (1984)		
A hypothetical workforce of 385 male smokers, 385 male nonsmokers, 115 female smokers, and 115 female nonsmokers	0.4 - 76	1.4 - 187.5
Advisory Committee on Asbestos[b] (1979a,b)		
Males and females	8.6 - 286	
Doll and Peto (1985)[c]		
Males	25.2	5.6

[a]Exposure of 25 years from age twenty-two.

[b]50 years exposure.

[c]Exposure of 35 years from age 20.

7.5 RELATIVE CARCINOGENICITY OF DIFFERENT FIBER TYPES

As briefly mentioned above, some differences exist among the major reports by different national organizations on the relative carcinogenicity of different asbestos fiber types. The view of the British in the Report of the Advisory Committee on Asbestos (1979a,b) and of Acheson and Gardner (1983), who wrote the background health effects paper and a 1983 update, is that crocidolite is a very potent mesothelial carcinogen, amosite is less so, and chrysotile rarely produces such a tumor. Their view is based on data similar to that of Table 3-35 and on the finding that in surveys of individuals with mesothelioma, particularly in Great Britain, an exposure to crocidolite or amphiboles can usually be documented either in a history or in analysis of lung tissue for asbestos fibers (a history of exposure to chrysotile is equally common). It is not certain how much weight one should place upon this latter evidence. In Great Britain, as in the United States, occupational exposure to asbestos largely involves exposure to mixtures of fibers. Thus, an association between amphibole exposure and mesothelioma would be expected. It is found that amphibole asbestos varieties are retained in the lung for decades after exposure, whereas chrysotile undergoes removal processes of various types. Thus, with even brief or low intensity amphibole exposures, fibers are commonly found in lung tissue analysis.

The Ontario Royal Commission (1984) also noted that there is a convincing case against amphiboles in relation to the incidence of mesothelioma and that, while chrysotile is capable of causing mesothelioma in humans, the incidence among chrysotile-exposed cohorts has been relatively low. For this, they cite the example of the Charleston, South Carolina textile plant with an extraordinarily high incidence of lung cancer, but only one mesothelioma.

Doll and Peto (1985) state that, in their opinion, the epidemiological data show that chrysotile can cause both mesothelioma and lung cancer but that peritoneal mesothelioma is rarely caused by chrysotile exposure and that crocidolite and amosite are more dangerous then chrysotile when used in the same way. Doll and Peto (1985) particularly noted the much greater mesothelioma risk in the experience of gas mask manufacturing workers who used crocidolite compared to those who used chrysotile (Acheson et al., 1982). However, no exposure data were available.

The view of the National Academy of Sciences (1984) report was that the epidemiological literature on the relative ability of different fiber types to

cause disease does not present a clear picture. The observed variation in risk may be due to different effects caused by different fiber types or dimensions used in processes in which other contaminants are present. They state that the magnitude of the difference in reported risks is not likely to be explained by fiber or process differences alone.

7.6 NON-MALIGNANT EFFECTS

All reviews of asbestos did not consider a non-malignant disease to be of importance at the exposures found in environmental circumstances. For example, the Ontario Royal Commission (1984) concluded that "at low levels of occupational exposure to asbestos the fibrotic process in the lungs, if indeed it can be initiated, will not likely progress to the point of clinical manifestation or even the mildest discomfort. On the basis of the available data our best judgement as to the lifetime occupational exposure to asbestos at which the fibrotic process cannot advance to the point of clinical manifestation of asbestosis is in the range of 25 f-y/mℓ and below."

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